Annals of Physical and Rehabilitation Medicine 59 (2016) 5–17

Available online at ScienceDirect www.sciencedirect.com

Elsevier Masson France EM|consulte www.em-consulte.com

Review

What are the disruptive symptoms of behavioral disorders after traumatic brain injury? A systematic review leading to recommendations for good practices

Angélique Stéfan *, Jean-François Mathé, the SOFMER group

Service de Médecine Physique et de Réadaptation Neurologique, Hôpital Saint-Jacques, CHU de Nantes, 85, rue Saint-Jacques, 44093 Nantes cedex, France

ARTICLE INFO

Article history:
Received 22 July 2015
Accepted 23 November 2015

Keywords:
Classification
Behavioral disorders
Brain injury
Good practices recommendations

ABSTRACT

Behavioral disorders are major sequelae of severe traumatic brain injury. Before considering care management of these disorders, and in the absence of a precise definition for TBI-related behavioral disorder, it is essential to refine, according to the data from the literature, incidence, prevalence, predictive factors of commonly admitted disruptive symptoms.

Methods: Systematic review of the literature targeting epidemiological data related to behavioral disorders after traumatic brain injury in order to elaborate good practice recommendations according to the methodology established by the French High Authority for Health.

Results: Two hundred and ninety-nine articles were identified. The responsibility of traumatic brain injury (TBI) in the onset of behavioral disorders is unequivocal. Globally, behavioral disorders are twice more frequent after TBI than orthopedic trauma without TBI (Masson et al., 1996). These disorders are classified into disruptive primary behaviors by excess (agitation 11–70%, aggression 25–39%, irritability 29–71%, alcohol abuse 7–26% drug abuse 2–20%), disruptive primary behaviors by default (apathy 20–71%), affective disorders – anxiety – psychosis (depression 12–76%, anxiety 0.8–24,5%, posttraumatic stress 11–18%, obsessive-compulsive disorders 1.2–30%, psychosis 0.7%), suicide attempts and suicide 1%.

Discussion: The improvement of care management for behavioral disorders goes through a first step of defining a common terminology. Four categories of posttraumatic behavioral clinical symptoms are defined: disruptive primary behaviors by excess, by default, affective disorders-psychosis-anxiety, suicide attempts and suicide. All these symptoms yield a higher prevalence than in the general population. They impact all of life's domains and are sustainable over time.

1. Introduction

Behavioral disorders are common and contribute to the severity of the trauma, around 62% at one year from trauma, regardless of the initial TBI severity [1]. These disorders are still present at 5 years posttrauma [2]. They frequently appear right from the awakening period and family members of patients with severe TBI report at 3 months post-TBI that patients "are not the way they used to be" in 49% of cases vs. 60% at one year and 74% at 5 years [3]. In this latter study by Brooks et al., the most frequently reported behavioral, affective or psychological changes concerned irritability (64%), bad temper (64%), tiredness (62%), depression (57%), rapid mood change (57%), anxiety (57%) and threat of violence (54%). At two years post-TBI, irritability was also reported as one of the most common problems by Ponsford et al. [4]. Furthermore, authors noted that lack of initiative was present in 44% of cases, and inappropriate social behavior in 26% of cases.

However, no systematic review of the literature was recently conducted. Furthermore, no precise definition of behavioral disorder post-TBI was found. In the population of persons with intellectual disabilities, Tassé et al. [5] defined serious behavioral disorders as "behaviors that are noxious for the health or physical integrity of the person". One can consider that a behavioral disorder exists when the behavior of a person is deemed deviant, unacceptable or dangerous compared to what would be considered normal within a group of person sharing the same values and a common culture.

The objective of this work was to refine the incidence, predictive factors and progression of the different behavioral disorders encountered after moderate or severe traumatic brain injury in adults. These recommendations were elaborated by a group of expert following the HAS protocol (http://www.has-sante.fr/portail/jcms/c_431294/recommandations-pour-la-pratique-clinique-rpc)

* Corresponding author. Tel.: +33 6 45 65 37 63; fax: +33 2 40 84 61 91.
E-mail address: stefan.angelique@wanadoo.fr (A. Stéfan).

http://dx.doi.org/10.1016/j.rehab.2015.11.002
1877-0657/

that includes several criteria of the PRISMA method (criteria 1, 2, 3, 6, 7, 13, 15) [6].

2. Methods

The steering group of this project, consisting of several experts in TBI care management, proposed to differentiate disruptive primary behaviors by excess from disruptive primary behaviors by default. Four subgroups of behavioral disorders were considered in this work:

- disruptive primary behaviors by excess;
- disruptive primary behaviors by default;
- affective disorders, anxiety and psychosis;
- suicide attempts and suicide.

Cognitive disorders were considered as a different entity and are not included in the framework of this article.

The review of the literature was conducted on Medline, in French and English from January 1990 to March 2012 as well as in books and articles not referenced in the Medline database, this search was conducted by the French High Authority for Health services. An additional search was conducted up to June 2015 without the help of the French High Authority for Health. Keywords used for the Medline search were the following: (“Craniocerebral Trauma” [Majr]) OR “Brain Injuries” [Majr] Or (Brain injur* Or Brain trauma* Or Head injur* Or Head trauma*) [title] AND “Mental Disorders” [Mesh] OR “Mood Disorders” [Mesh] OR “Anxiety” [Mesh] OR “Anxiety Disorders” [Mesh] OR “Depression” [Mesh] OR “Depressive Disorder” [Mesh] OR “Depressive Disorder, Major” [Mesh] OR “Psychotic Disorders” [Mesh] OR “Apathy” [Mesh] OR “Aggression” [Mesh] OR “Irritable Mood” [Mesh] OR “Anger” [Mesh] OR “Psychomotor Agitation” [Mesh] OR “Substance-Related Disorders” [Mesh] OR “Cognition Disorders” [Mesh] OR “Executive Function” [Mesh] OR “Awareness” [Mesh] OR “Agnosia” [Mesh] AND “Epidemiology” [Mesh] OR “Prevalence” [Mesh] OR “Incidence” [Mesh] NOT “Critical Care” [Mesh] OR “Child” [Mesh] OR “Infant” [Mesh]OR “Pediatrics” [Mesh] OR “Adolescent” [Mesh] Or (Critical care OR child* OR infan* Or paediatr* or pediatr* OR adolescent*).

Articles focusing on the prevalence or incidence of all types of behavioral disorders post-TBI were kept for this analysis (follow-up of cohorts, cross-sectional studies, longitudinal studies, case studies). Studies that had a more general approach on neuropsychiatric disorders post-TBI were also considered. Articles related to evaluation scales were not kept for this analysis, they are the focus of another article in this special thematic issue. Studies targeting other pathologies than moderate to severe TBI (other neurological diseases, mild TBI, TBI in war veterans) were excluded. Articles were analyzed taking into account potential biases (selection bias, heterogeneity of the population, inclusion delay, use of specific adapted scales).

3. Results

The electronic searches (Medline 1990–2012) returned 162 citations and 42 in the additional research (Medline 2012–2015). After screening titles and abstracts, 124 records were discarded as irrelevant or obviously not meeting the selection criteria. Hand-searching identified 219 additional eligible articles, hence leaving a total of 299 articles.

3.1. Disruptive primary behaviors by excess

The analysis of the literature unveiled several symptoms such as agitation, opposition, wandering behaviors, disinhibition, irritability, impulsivity, screams and shouting, risk-taking attitudes, bulimia, addictions, hypersexuality, exhibitionism, Kluver-Bucy* syndrome, hostility, aggression, verbal and physical violence. Some themes such as opposition, aggression, circadian rhythm disorders, inappropriate wandering or motor behaviors, screams, motor disinhibition are symptoms similar to the ones present in Alzheimer’s disease and answer to the same characteristics (good practices recommendations: Alzheimer’s disease and related disorders: management of disruptive behavioral disorders – HAS website 2009). It was decided to classify the disorders into 5 subchapters:

- agitation;
- aggression;
- irritability;
- substance abuse: at-risk, excessive, dependent behavior;
- behavior with medicolegal consequences, felony and crime.

3.1.1. Posttraumatic agitation

Posttraumatic agitation is a common if not unavoidable characteristic of the coma-awakening period [7]. It is related to the altered state of consciousness [8] posttraumatic amnesia period [9], and decreases when cognitive functions improve [10]. The duration is usually short (1 to 14 days) but can sometimes last longer or appear later on [10,11]. No type of behavior defines agitation, it can be a combination of aggression, akathisia, disinhibition, emotional lability, motor restlessness [9] or for others, impulsivity, disorganized thinking, perceptual disturbances, impaired capacity to sustain attention or reduced adaptation [12]. The mean incidence of agitation is estimated at 46% with ranges going from 11 to 70%. Environmental causes, sleep disorders, pain promote agitation [13]. The functional future is related to the duration and severity of the agitation [14] (see Table 1: main studies on agitation after TBI).

3.1.2. Aggression

Aggression includes verbal aggression, physical aggression against objects, physical aggression against self, other persons [19] but also severe irritability, violent, hostile, or assaultive behavior and “episodic dyscontrol” [20]. After traumatic brain injury, hostile or explosive aggression is more frequent than goal-directed aggression [21]. Aggression incidence varies between 25 and 39%. It is related to the severity of the initial trauma [22] and the existence of a prefrontal injury (orbitofrontal) [23]. Aggressive behaviors are more frequent in older male subjects, when there is associated language disorders, in a noisy environment, in the 24 hours following epileptic seizure [24]. Depression and anxiety are more common in the aggressive TBI patient [23,25]. Anger is more frequent in patients with executive function disorders [26] (see Table 2: main studies on aggression after TBI). More recently, a link between history of aggression and verbal aggression post-TBI was evidenced [27,28].

3.1.3. Irritability

Irritability can be defined as an excessive reaction with unjustified anger fits. Its incidence ranges from 29 to 71% according to studies in patients with severe TBI. Risk factors of an irritable behavior in patients with TBI are: being male, age between 15 and 34, unemployment, social isolation, depression [36,37]. Contrarily to irritability occurring after mild TBI, authors reported the absence of a correlation between cognitive impairment and irritability after severe TBI [38].

3.1.4. Addictions with abuse and excesses

If addictions with abuse and excesses of alcohol or illicit substances are problematic in the care pathway after TBI, the

Table 1
Main studies on prevalence rates of agitation after traumatic brain injury.

References	Design	Level	Conclusion
Levin and Grossman, 1978 [10]	Longitudinal study, $n = 80$ closed head injury of graded severity	4	Agitation: 35%
Reyes et al., 1981 [11]	Longitudinal study, $n = 87$ traumatic head injury followed over 5 years or more from admission to post-discharge	4	Agitation: 50%
Brooke et al., 1992 [15]	Longitudinal Study, $n = 100$ severe closed head injury admitted to a regional level I Trauma Center with a Glasgow Coma Scale < 8 + more than one hour of coma + more than one week of hospitalization. OAS	4	Aggression: 11% 11% exhibited episodic agitation. Eight subjects were agitated for one week, one for two weeks, one for three weeks, and one for four weeks Restlessness: 35%
Bogner and Corrigan, 1995 [8]	100 consecutive patients admitted in rehabilitation	4	42% demonstrated agitation
Wolf et al., 1996 [16]	Survey study. All skilled nursing facilities in the state of Connecticut (253)	4	One hundred and sixty-two, or 64%, responded to the survey, 39 (24%) of the facilities reported 140 individuals with a primary diagnosis of brain injury, 45% of the 39 facilities had brain-injured patients with agitation
Bogner et al., 2001 [17]	Prospective longitudinal study, $n = 340$ consecutive TBI admitted in an acute brain injury rehabilitation. Age ≥ 14 years. Severe TBI 64%, moderate TBI 13%, mild TBI 22%. Assessment with ABS	4	36% of agitation Agitation was associated with: longer length of stay and decreased functional independence in the cognitive realm at discharge
Nott et al., 2006 [18]	Retrospective study, $n = 80$ TBI (graded severity) admitted for rehabilitation	4	70% of patients demonstrated agitation during rehabilitation for an average of 32 days (86% in acute stage of rehabilitation) Agitated patients experienced increased length of stay, longer PTA duration, reduced functional independence at discharge

GCS: Glasgow Coma Score; OAS: Overt Aggression Scale; ABS: Agitated Behaviour Scale.

Table 2
Main studies on prevalence rates of aggression after traumatic brain injury.

References	Design	Level	Conclusion
Tateno et al., 2003 [23]	Case-control study: 89 severe TBI matched 26 multiple trauma cases (without TBI) Assessment with the OAS in the 6 months after injury	3	33.7% of the TBI group met the criteria for aggressive behavior compared with 11.5% of the non-TBI injured Major depressive disorder, history of alcohol and substance abuse, poor premorbid social functioning, frontal lobe lesion was more frequent in the aggressive group
Galski et al., 1994 [29]	Prospective study, $n = 13$ TBI	4	Aggression: 39%: 29% physical aggression; 21% verbal aggression; 11% both
Johnson and Balleny, 1996 [30]	Prospective study: $n = 33$ severe TBI. Short questionnaire using seven categories of behavior including physical and verbal aggression	4	6% in hospital, 13% 18 months or less since injury, 55% more than 18 months since injury (family report)
Alderman et al., 1997 [31]	Prospective study, $n = 18$ severe TBI. Assessment with OAS-MNR	4	15 TBI have 76 aggressive behavior
Kant et al., 1998 [32]	Longitudinal Prospective study: $n = 13$ closed head injury with aggression (mild TBI 5, moderate TBI 6, severe TBI 2). Assessment with OAS-M	4	OAS-M mean score for 13 TBI = 230.54 This study does not calculate incidence or prevalence
Baguley et al., 2006 [25]	Retrospective study: 228 patients with moderate to severe TBI (post-discharge). Assessment with OAS at 6 months ($n = 149$), at 24 months ($n = 133$), at 60 months ($n = 60$)	4	25% of aggression at 6 months at any given follow-up period (but participants differ) Aggression was consistently associated with depression concurrent traumatic complaints, younger age at injury, and low satisfaction with life
Alderman, 2007 [24]	Prospective study. Records of aggressive behaviour shown by 108 patients over 14 days Assessment with OAS-MNR	4	729 physical assaults were made on others
Giles and Mohr, 2007 [33]	Inter-rater reliability of an incident-based rating scale for aggressive behaviour following traumatic brain injury $n = 17$, TBI. Assessment with OAS-MNR-E	4	There were 199 observed aggressive behaviors: verbal aggression: 66, physical aggression against objects: 33, physical aggression against self: 7, and physical aggression against others: 97
Rao et al., 2009 [34]	Observational prospective study of the prevalence of aggression in the 3 months following TBI in a cohort of participants recruited within 3 months of trauma. $n = 67$, TBI. Assessment with OAS	4	Prevalence of aggression was found to be 28.4%, predominantly verbal aggression Post-TBI aggression was associated with new-onset major depression, poorer social functioning, and increased dependency in activities of daily living
Dickens et al., 2011 [35]	Prospective study about OAS-MNR and Attacks scales to measure aggression during 6 weeks on a brain injury unit. $n = 40$ patients in National Brain Injury Center	4	82.5% had demonstrated physical aggression against people during the 6-week period. The total number of incidents logged on the OAS-MNR was 1066. Two patients were involved in 841 (78.9%) of the incidents: severe aggression: 6%; moderate aggression: 66%; mild aggression: 26%

OAS: Overt Aggression Scale; OAS-MNR-E: Overt Aggression Scale-Modified for Neurorehabilitation-Extended.

review of the literature showed that TBI did not induce more substance use after the trauma. The prevalence of alcohol or illicit substance abuse is even lower in the year following TBI [39]. The use of tools based on self-assessment of the disorder by the patient is a probable confusion factor. Only rare studies have shown that alcohol consumption could worsen 2 to 5 years after TBI with a return to prior level of consumption before the trauma [40]. However, if data from the literature are not univocal, experts consider that the risk of harmful use or dependence appear more important at a distance from TBI and especially after returning home post-discharge [41]. Factors related to the detrimental use of substances or alcohol or substance/alcohol dependence are: being male, low educational attainment and low social level, depression. Drug or alcohol use before TBI could increase the risk of behavioral disorders after TBI, risk of dependence, depression and suicide [42].

We did not find any specific article on the use and/or abuse of cocaine, psycho-stimulants or opioids. Eating disorders are often described after TBI, but the review of the literature is quite scarce, limited most often to case studies [43]. These symptoms, especially bulimia can be intertwined with other behavioral disruptions such as the Kluver-Bucy syndrome [44] or endocrine disorders, common after TBI [45].

3.1.5. Behavior with medicolegal consequences

The issue of a relationship between TBI and a behavior with medicolegal consequences is often brought up. Several epidemiological studies, essentially American ones, looked for the prevalence of TBI history in incarcerated populations. The prevalence of TBI history was significantly higher in inmates than the prevalence estimated in the general population (meta-analyses by Farrer in 2011 and Shiroma in 2010, focusing on around 5000 inmates each) [46,47]. The prevalence of TBI ranges from 9.7% to 100% according to the studies. Shiroma [47], found a 60.2% TBI prevalence in prisoners. Inmates with a history of TBI tend to be younger, with more health problems, more memory disorders, have longer incarceration periods, are more frequently incarcerated, more likely to have a substance abuse problem (alcohol or marijuana) and more anxiety-depression disorders than other inmates without TBI. If TBI occurs during childhood, it is associated with an increased risk of psychiatric disorders and criminality. Most studies are based on the self-reported presence of TBI. The absence of a control group and variability were other biases limiting the methodological quality of these studies.

3.2. Disruptive primary behaviors by default

Symptoms by default observed after TBI include apathy, apragmatism, athymhormia and abulia.

3.2.1. Apathy

Apathy is the most commonly studied behavior by default post-TBI [48]. For Marin [49], it is characterized by a reduction of goal-targeted behaviors and involves motivational, emotional and/or cognitive aspects whether those are spontaneous behaviors and/or in reaction to external stimuli. Levy and Dubois [50] defined three subtypes of disrupted processes: 'emotional-affective', 'cognitive' and 'auto-activation'. Apathy diagnostic criteria proposed by the European Psychiatric Association, the French Association of Biological Psychiatry and the European Alzheimer's Disease Consortium [51,52] all report the criterion of decreased motivation, characterized by three symptoms:

- loss or decrease of goal-directed behaviors;
- loss or decrease of goal-directed cognitive activity;
- absence or decrease of emotions.

New models are being developed (see Arnould et al. in this issue). Its prevalence varies between 20 and 71% according to the different studies on patients with severe TBI. The association between apathy and depression is common but apathy can exist without depression and vice-versa, which suggests the implementation of distinct processes [53]. If there is no specific scale to differentiate depression from apathy, the richness of the affects mobilized can help make the distinction. Some traits like anhedonia, social isolation, poorly organized speech and blunted affect are higher after TBI [54]. Apathy can occur at a distance from the initial trauma. It is associated with dysexecutive syndrome [55] correlated to the performances on the Behavioural Assessment of the Dysexecutive Syndrome (BADS) scale [56] as well as emotional and motivational dysfunctions observed by neurovegetative symptoms with an inverse correlation between apathy and heart rate reactivity to stress [57]. This symptom impairs rehabilitation, has an impact on autonomy at home, professional future and burden felt by families (see Table 3: main studies on apathy after TBI).

3.3. Affect disorders, depression, anxiety and psychosis

3.3.1. Depression

Depression is one of the most common psychiatric complications after TBI [61]. There is no specificity regarding diagnostic criteria of depression in patients with TBI (see DSM 5) but anosognosia, communication disorders can interfere with the patient's mood and render the diagnosis more difficult. A review of the literature conducted in 2010 by the American Health Agency identified 115 publications on this topic. The mean prevalence was reported at 30% after moderate to severe TBI (12 to 76%) [62], i.e. 7.5 times greater than in the general population [63], even when excluding analyses focusing on veterans, mild traumatic brain injury, studies not refining the severity of the trauma. Prevalence does not vary much over time, and affects about a third of patients at 3 months to a year post-TBI. Fatigue, distraction, sleep disorders are common in TBI patients in the absence of depression, which might be confusing for physicians and might lead them to overestimate the diagnosis. The risk of developing major depression after TBI is reinforced by a prior psychiatric pathology and low socio-economic status [64]. There are numerous factors associated with depression: being a woman, being young, prior psychiatric pathology, depression at the time of the accident, lesions of the left hemisphere (dorsolateral prefrontal cortex and/or basal ganglia) [65], higher cognitive deficits, social aggression, unemployment, fear of losing one's job or absence of a job, social isolation, low socio-economic status, substance abuse, perceived stress, litigation, pain, self-awareness of the severity of the disability. Anxiety disorders are most frequently associated with depression (31 to 61% of cases) with a reported 37% frequency rate for the association of posttraumatic stress and depression. The expression of depressive symptoms is lower in patients with anosognosia. Family support, psychosocial caregivers, availability of a confident, direct return home all have a protective effect against depression [66]. Anxiety-related depression might be related to damages to the right hemisphere [65] (see Table 4: main studies on depression after TBI).

3.3.2. Anxiety symptoms

Anxiety symptoms are presented into 10 groups according to the DSM-IV: agoraphobia, panic attack, social phobia, OCD, posttraumatic disorder, acute stress syndrome, generalized anxiety, anxiety disorder linked to a medical pathology, substance-induced anxiety disorder. The risk of presenting with generalized anxiety after TBI is 2.3 times higher than in the general population, i.e. 9.1% [102], panic attack is 5.8% more common than in the

Table 3
Main studies on prevalence rates of apathy after traumatic brain injury.

References	Design	Level	Conclusion
Andersson et al., 1999 [57]	72 consecutive in-patients (28 TBI, 30 stroke and 14 hypoxic brain injury) Rehabilitation unit in Norway Assessment with AES-C ≥ 34	3	Apathy after TBI: 46%, after stroke: 56%, after hypoxic brain injury: 78% AES score is higher after hypoxic brain injury, subcortical damage, right hemispheric damage
Rao et al., 2007 [54]	Case-control study: 12 patients with apathy matched with16 people with schizophrenia Assessment with SANS	3	Severe TBI: 50%, moderate TBI: 33.3%, mild TBI: 16% SANS: patients with schizophrenia had more severe anhedonia, blunted affect, and alogia
Andersson and Bergedalen, 2002 [55]	$n = 53$ severe TBI Assessment with AES-C (cut-off > 34)	4	Time since TBI: 12.2 months (± 10.06) Apathy: 62.3%
Al-Adawi et al., 2004 [58]	80 TBI (6 mild, 2 moderate, 36 severe) Assessment with AES-S (cut-off > 34)	4	Time since TBI: 8.35 months (± 4.50) Apathy: 20%
Kant et al., 1998 [53]	Prevalence study, $n = 83$ TBI seen in a neuropsychiatric clinic. (Severe TBI + moderate TBI: 20.5%) Assessment with AES-S ≥ 34 and BDI > 11	4	Apathy: 71% with or without depression (59 patients) Group 1: apathy without depression: 10.8% (9 patients) (AES-S: 40.5 ± 6.26) Group 2: depression without apathy: 10.8% (9 patients) (BDI: 18 ± 5.26) Group 3: both depression and apathy: 60.2% (50 patients): (AES-S: 43.92 ± 7.56 and BDI: 23.5 ± 8.58) Group 4: neither depression nor apathy: 18.1% (15 TBI) Younger patients are more likely to be apathetic Older patients are more likely to be depressed Patients with severe injury were more likely to exhibit apathy alone
Lane-brown and Tate, 2009 [59]	Cross-sectional study $n = 34$ severe TBI (> 6 months since TBI) Assessment with AES	4	Apathy alone: 10 patients Apathy and depression: 11 patients Depression without apathy: 4 patients
Ciurli et al., 2011 [60]	$n = 120$ TBI Assessment with NPI	4	Time since TBI: 106 months (± 15.1) Apathy: 42%

AES-S: Apathy Evaluation Scale–version patient; AES-C: Apathy Evaluation Scale – version clinician; SANS: Scale for the Assessment of the Negative Symptoms; BDI: Beck Depression Inventory; NPI: Neuropsychiatry Inventory.

general population, i.e. 9.2%. Generalized anxiety disorder has been correlated with a poor functional status [80].

3.3.3. Posttraumatic stress disorder (PTSD)

The frequency of posttraumatic stress disorder (PTSD) after severe TBI was estimated at between 11 and 18% of cases, up to 53% of cases as reported in a recent German study [103]. PTSD sometimes sets in the immediate TBI aftermath, yet most often it occurs after a few days or weeks. Its existence has often been considered as improbable because of posttraumatic amnesia (PTA). One can explain it by the presence of "islands of memory" during PTA, unconscious cognitive traces of the trauma, reconstructed memories (pseudo-memory), and/or traumatic experiences related to the event and experienced at a certain distance (painful medical treatment, stressful perceptions when coming out of the PTA phase). Some PTSD symptoms are common after TBI independently of stress (sleep disorders, irritability, memory and concentration disorders, decreased thought-processing speed, fatigue, depression, headaches, etc.). Its progression seems to be slow but favorable [104–106].

Sometimes one can observe a shift to a sustainable change in personality or depressive state [64].

3.3.4. Obsessive-compulsive disorders (OCD)

The incidence of obsessive-compulsive disorders (OCD) is greater than in the general population (relative risk estimated at 2.6) with an incidence ranging between 1.2 and 30% [102,107]. Damages to the orbitofrontal cortex, cingulate cortex and subcortical structures (caudate nucleus) seem to promote OCD [108]. Symptoms can appear quickly after the initial trauma (from a few hours to a week) [80] (see Table 5: main studies on the prevalence of anxiety, posttraumatic stress disorder, and obsessive-compulsive disorder post-TBI).

3.3.5. Psychosis

There is limited evidence of an association between psychosis and severe or moderate TBI [113]. In a meta-analysis from 2011 grouping 172 studies, Molloy et al. [114] compared the risk of "schizophrenia" in persons with TBI vs. the risk of "schizophrenia" in a control group. There is a significant link between TBI and schizophrenia (OR = 1.65; 95% CI = 1.17–2.32) but with an heterogeneity of the studies, especially concerning the definition of "schizophrenia", sometimes described as "schizophrenia", "psychosis", "psychotic disorder", "delirium disorder", etc. The risk of developing psychosis after moderate or severe TBI becomes significant in the second year post-TBI (OR = 5.9; 95% CI = 1.6–22.1) and appears to remain significant the third year post-TBI (OR = 3.6; 95% CI = 1.0–12.3). However, since age at the onset of schizophrenia (15 to 25 years of age) corresponds to the peak of frequency of TBI, it is often difficult to exclude the development of psychosis independently from TBI. Did the psychotic disorder exist prior to the TBI? Did TBI play a facilitating role or trigger a causal relationship? The incidence varies between 0.1 and 9.8% [111], prevalence is estimated at 0.7% [63]. Symptoms consist of paranoid illusions with persecution themes (55%), grandiosity (20%), hallucinations (most often auditory ones), aggression (40% of cases) [115]. Negative symptoms, catatonia, thought disorders are not common. The risk of developing psychosis increases when there is a family history of psychosis, when TBI occurs during adolescence, in case of temporal lesions and when TBI is severe [116]. In 2008, Kim [117] concluded his literature review by underlining the importance of differentiating secondary psychotic disorders from schizophrenia. TBI increases the risk of schizophrenia in persons genetically predisposed to the disease due to a synergic interaction between genes and the neurological trauma [117]. The definition of psychosis varies in the literature and definition criteria lack clarity, thus conclusions are hard to draw

Table 4
Main studies on prevalence rates of depression after traumatic brain injury.

Reference	Design	Level	Conclusion
Bombardier et al., 2010 [67]	Prospective cohort, $n = 559$ TBI ($n = 1080$ eligible) US study in Trauma Center Assessment with PHQ9	2	Prevalence of depression was 31.1% at 1 month; 24.7% at 2 months; 24.5% at 3 months; 20.8% at 6 months; 24.2% at 8 months; 27.1% at 10 months; 23. 3% at 12 months
Hart et al., 2011 [68]	Prospective cohort, $n = 1570$ TBI US Study, in Trauma Center Assessment with PHQ9	2	Prevalence of depression > 12 months since TBI was: 22% of minor depression; 26% of major depression
Dunlop et al., 1991 [69]	Prospective study, $n = 68$ TBI US study Assessment with NRS	3	Prevalence of depression 3 to 6 months since injury was 50%; 28% 6 to 12 months
McCleary et al., 1998 [70]	Case-control study, $n = 105$ TBI matched with 40 individuals US study in tertiary care center Assessment with SCL90 et NRS	3	Prevalence of depression 6 to 12 months since TBI was 24.4% (SCL90); 33% (NRS) Prevalence of depression > 12 months was 20% (SCL90); 33% (NRS)
Curran et al., 2000 [71]	Case-control study, $n = 88$ TBI matched with 61 patients with orthopedic injuries Australia, rehabilitation center Assessment with BDI	3	Prevalence of depression was 55.7%
Fann et al., 2004 [72]	Case-control study TBI/non-TBI, $n = 691$ Assessment with SCID	3	Prevalence of major depression was 33%
Jorge et al., 2004 [73]	Case-control study $n = 91$ TBI (mean GCS = 12.3)/ matched with $n = 27$ trauma patients without central nervous system damage. US study Assessment with SCID	3	Prevalence of depression was 20.2%; 32.4%, 3 to 6 months since injury; 40.5%, 6 to 12 months since injury
Popovic et al., 2004 [74]	Case-control study, $n = 67$ TBI/78 healthy patients Serbian in Tertiary care center Assessment with ZDS	3	Prevalence of depression > 12 months since TBI was 46.3%
Frenisy et al., 2006 [75]	Case-control study, $n = 25$ TBI (GCS mean = 12.5) versus 25 multiple trauma cases French study in Tertiary care center Assessment with NRS-R, and SCL90-R	3	Prevalence of depression > 12 months since TBI was 76%
Gagnon et al., 2006 [76]	Case-control studies, $n = 30$ severe or moderate TBI matched with 30 participants without TBI Canadian study in Rehabilitation center Assessment with BDI	3	Prevalence of depression > 12 months since TBI was 50%
Ziino and Ponsford, 2006 [77]	Case-control study, $n = 46$ TBI Australian study Assessment with HADS	3	Prevalence of depression > 12 months since TBI was 39.1%
Hawthorne et al., 2009 [78]	Case-control studies. $n = 66$ TBI/66 participants without TBI Canadian Study in Trauma Center Assessment with HADS	3	Prevalence of depression > 12 months since TBI was 22.7%
Varney et al., 1987 [79]	$n = 120$ closed TBI Assessment with DSM-III	4	Prevalence of depression was 76.7%
Jorge et al., 1993 [65]	Cross-sectional study. $n = 66$ TBI US study in Trauma center Assessment with SCID	4	Prevalence of depression was 28.8%; 29.6% 3 to 6 months since injury; 25.6% 6 to 12 months; 25.6% > 12 months
Fann et al., 1995 [80]	Cross-sectional study, $n = 50$ TBI US study in Rehabilitation center Assessment with SCID	4	Prevalence of depression > 12 months since TBI was 54%
Gomez-Hernandez et al., 1997 [81]	Longitudinal study + cross-sectional, $n = 65$ TBI Spain study Assessment with SCID	4	Prevalence of depression was 35.4%; 37.5% 3 to 6 months since injury; 38.1% 6 to 12 months; 27% > 12 months since injury since injury
Kant et al., 1998 [53]	Cross-sectional study, $n = 83$ TBI US study in psychiatric center Assessment with BDI and AES	4	Prevalence of depression was 11% (BDI) and 60% (AES)
Deb et al., 1999 [82]	Cross-sectional study, $n = 164$ TBI including 30 TBIM/S UK study in Tertiary care center Assessment with SCAN and Behavior Checklist	4	Prevalence of depression > 12 months since TBI was 12.8% (SCAN); 19.5% (behavior checklist)
Sherman et al., 2000 [83]	Cross-sectional study, $n = 175$ TBI Canadian study Assessment with MMPI	4	Prevalence of depression > 12 months since TBI was 33% for all severity 36% for severe and moderate TBI
Van Reekum et al., 2000 [63]	Review	-	Prevalence of major depression was 44.3%
Bryant et al., 2001 [84]	Cross-sectional study, $n = 96$ TBI (mean GCS: 8) Australian study in Rehabilitation center Assessment with BDI	4	Prevalence of depression > 12 months since TBI was 45.8%
Kersel et al., 2001 [85]	Longitudinal study, $n = 58$ TBI severe (123 eligible) New Zealand study in Tertiary care center Assessment with BDI	4	Prevalence of depression 6 to 12 months since injury was 24% 24.1% > 12 months since injury
Rapoport et al., 2002 [22]	Cross-sectional study, $n = 282$ TBI (323 eligible with139 severe TBI) Canadian study in Tertiary care center Assessment with NRS (NR)	4	Prevalence of depression was 34.3% Prevalence of major depression 3 to 6 months since injury was 48.9%

Table 4 (*Continued*)

Reference	Design	Level	Conclusion
Seel et al., 2003 [86]	$n = 666$ TBI (GCS = 8.6) US study in Rehabilitation center Assessment with NFI-D	4	Prevalence of depression > 12 months since TBI was 27%
Ashman et al., 2004 [87]	Cross-sectional and longitudinal study, $n = 188$ TBI US study in Tertiary care center. Assessment with SCID	4	Prevalence of depression was 35% (T1 = 3 months–4 years), 24% 12 months after T1, 21% 24 months after T1
Franulic et al., 2004 [88]	Cross-sectional study, $n = 71$ TBI Chilean study in Tertiary care center Assessment with HAMD	4	Prevalence of depression > 12 months since TBI was 42.3%
Evans et al., 2005 [89]	Cross-sectional study, $n = 96$ TBI/135 US study in rehabilitation center. Assessment with CES-D	4	Prevalence of depression was 54%
Huang et al., 2005 [90]	Cross-sectional study $n = 59$ TBI including 17 severe cases US study in Rehabilitation center Assessment with SCID and ZDS	4	Prevalence of depression > 12 months since injury was (SCID) 13.6% (41% for severe TBI), 16.9% (ZDS) (59% for severe TBI)
Kennedy et al., 2005 [91]	Cross-sectional study, $n = 78$ TBI (mean GCS = 9.3) (severe = 43%) US study Assessment with SCID	4	Prevalence of depression > 12 months since TBI was 30%
Chiu et al., 2006 [92]	Cross-sectional study, $n = 199$ TBI (7.5% severe) Taiwan study in Tertiary care center Assessment with CES-D	4	Prevalence of depression > 12 months since TBI was 23.9%
Rapoport et al., 2006 [93]	$n = 69$, Assessment with SCID	4	Prevalence of depression was 12.2%
Al-Adawi et al., 2007 [95]	Cross-sectional study, $n = 68$ TBI Oman study in Tertiary care center. Assessment with CIDI and HADS	4	Prevalence of depression > 12 months since TBI was 57.4% (CIDI), 19.1% (HADS)
Kim, 2007 [63]	Review	–	Prevalence of depression was 15.6 to 61%
Sherer et al., 2007 [94]	Cross-sectional study, $n = 49$ severe TBI (69 eligible) US study in Rehabilitation unit Assessment with CES-D	4	Prevalence of depression was 31.9%
Hawley and Joseph, 2008 [96]	Cross-sectional study, $n = 165$ TBI (563 eligible/103 severe) UK study. In rehabilitation center Assessment with HADS	4	Prevalence of depression was 20.5% (17.2% for severe TBI) 6 to 12 months since TBI
Whelan-Goodinson et al., 2008 [97]	Cross-sectional study, $n = 100$ TBI Australian study in Tertiary care center Assessment with SCID	4	Prevalence of depression was 34% > 12 months since TBI
Fann et al., 2009 [98]	Cross-sectional study, $n = 145$ TBI (19.3% severe TBI) US study in Trauma center Assessment with PHQ-9	4	Prevalence of depression was 25.5%
Peleg et al., 2009 [99]	Cross-sectional study, 65 patients TBI Israeli study in Rehabilitation center Assessment with BDI	4	Prevalence of depression was 73.9% > 12 months since TBI
Rao et al., 2009 [34]	Cross-sectional study, $n = 67$ TBI (107 eligible) US study in Rehabilitation unit Assessment with SCID	4	Prevalence of depression was 11.9%
Ponsford and Schönberger, 2010 [100]	Cross-sectional study, $n = 301$ TBI Australian study in Tertiary care center Assessment with HADS; 266 patients at 60 months	4	Prevalence of depression was 45% at 24 months 44% at 60 months
Seel et al., 2010 [101]	$n = 666$ moderate and severe TBI	4	Fatigue: 29% Attention and concentration disturbances: 28% Anger and irritability: 28% Ruminations: 25% 27% major depressive episode

SCID: Structured Clinical Interview for DSM-IV; CIDI: Clinical Depression/anxiety determined by Clinical Interview; HADS: Hospital Anxiety and Depression Scale; PHQ9: Patient Health Questionnaire; BDI: Beck Depression Inventory; CES-D: Center for Epidemiologic Studies Depression Scale; SCAN: Schedule for Clinical Assessment in Neuropsychiatry; NRS: Neurobehavioral Rating Scale; HAMD: Hamilton Depression Rating Scale; SCL-90: Symptom Checklist; ZDS: Zung Self Rating Depression Scale; AES: Apathy Evaluation Scale; MMPI: Minnesota Multiphasic Personality Inventory; NFI: Neurobehavioral Functioning Inventory.

(see Table 6: main studies on prevalence of psychosis after traumatic brain injury).

3.4. Suicide attempts and suicide

3.4.1. The rate for suicide, suicide attempts and suicide ideation

The rate for suicide, suicide attempts and suicide ideation is higher in the population of persons with TBI compared to the general population even after checking for psychiatric disorders [125,126]. The relative risk of suicide in case of severe TBI is evaluated at 3–4 times higher than the general population [41,127] with a cumulated suicide rate of 1% in the 15 years post-TBI. Suicide ideation is observed in 21–22% in patients with TBI [128–130] and has been associated to a higher relative risk of suicide attempts (OR 4.9; 95% CI 1.79–13.17). It is more frequent in cases of depression, anxiety and posttraumatic stress disorder [131]. Suicide attempts are hard to quantify but could reach 18% [128]. Evidence of a relationship between TBI and suicide attempts

Table 5
Main studies on prevalence rates of anxiety, posttraumatic stress disorder, and obsessive-compulsive disorder after traumatic brain injury.

References	Design	Level	Conclusion
Fann et al., 1995 [80]	$n = 50$ TBI Measured by 'Medical Outcomes Study Health Survey'	4	Prevalence of Generalized Anxiety Disorder: 24%
Hibbard et al., 1998 [109]	$n = 100$ TBI DSM-IV as measured by SCID	4	19% Posttraumatic Stress Disorder 14% Panic Disorder 10% phobia 15% Obsessive-Compulsive Disorder
Deb et al., 1999 [82]	$n = 120$ TBI measured by 'Clinical Interview Schedule Revised' and 'Psychosis screening Questionnaire'	4	9% panic disorder 2.5% Generalized Anxiety Disorder 0.8% phobia 1.6% Obsessive-Compulsive Disorder
Van Reekum et al., 2000 [63]	Review	–	9.1% Generalized Anxiety Disorder (relative risk = 2.3) 9.2% Panic Disorder (relative risk = 5.8)
Hoofien et al., 2001 [110]	$n = 76$ severe TBI followed in rehabilitation center. DSM-III	4	44% Generalized Anxiety Disorder at 15 years 30% Obsessive-Compulsive Disorder in the 15 years post-TBI
Silver et al., 2001 [111]	$n = 361$ outpatients, moderate and severe TBI Assessment with DSM-III	4	3.3% Panic Disorder 4.7% Obsessive-Compulsive Disorders
Koponen et al., 2002 [112]	Retrospective study, $n = 60$ TBI, assessed 30 years after TBI Assessment with DSM-IV	4	1.7% Generalized Anxiety Disorder 8.3% Panic Disorders

TBI: Traumatic Brain Injury; DSM: Diagnostic and Statistical Manual of Mental Disorders; SCID: Structured Clinical Interview for DSM-IV Diagnoses.

remains insufficient [113] (see Table 7: main studies on the prevalence of suicides and suicide attempts after TBI).

4. Discussion

The first recommendation drawn by the SOFMER working group concerned the need for all actors, healthcare professionals, medicosocial professionals, judges, lawyers... to share the same definitions and a common nomenclature for behavioral disorders (expert opinion). In the absence of consensus on the classification of these disorders, but according to experts of the steering group and the frequency of onset of symptoms reported in the literature, the choice was made to classify the symptoms in 4 subgroups. Disorders are classified in disruptive primary behaviors by excess

Table 6
Main studies on prevalence rates of psychosis after traumatic brain injury.

Référence	Design	Level	Conclusion
Silver et al., 2001 [111]	Cohort study: Prevalence rates of neuropsychiatric disorder in individuals with and without a history of traumatic brain injury (New Haven NIMH Epidemiological Catchment Area Study), $n = 361$ TBI among 5037 individuals	2	Risk estimate for psychosis following TBI: OR 1.80 (1.0–3.3)
Malaspina et al., 2001 [118]	Family study, $n = 1931$ TBI in the register "National institute of mental genetics initiative for schizophrenia and bipolar disorders"	2	Risk estimate for psychosis following TBI: OR 3.32 (1.77–6.22)
Timonen et al., 2002 [119]	Northern Finland 1966 Birth Cohort Study $n = 10934$	2	Risk estimate for psychosis following TBI: OR 1.10 (0.41–2.96)
Fann et al., 2004 [72]	Cohort study, $n = 939$ patients on "Adult Health Maintenance"	2	Risk estimate for psychosis following TBI: OR 3.60 (1.00–12.30)
Harrison et al., 2006 [120]	Cohort study, $n = 47754$ Swedish men and women born between January 1973 and December 1980	2	Risk estimate for psychosis following TBI: OR 1.10 (0.82–1.47)
Sachdev et al., 2001 [121]	Case-control study, $n = 45$ TBI with psychosis matched with $n = 45$ TBI without psychosis	3	The mean age of onset of psychosis is 26.3 years The mean latency is 54.7 months between injury and onset of psychosis with a gradual onset and subacute or chronic course Symptoms are paranoid delusions and auditory hallucinations (in 55% of patients) with delusion of persecution or grandiosity (20%). Negative symptoms (formal thought disorder, catatonic features) are uncommon
Nielsen et al., 2002 [122]	Case-control study. Rates of head injury among 8288 persons in the 15 years up to their first admission with schizophrenia and compared with 82,880 age- and gender-matched controls	3	Risk Estimate for Psychosis Following TBI: OR 0.89 (0.76–1.04)
Abdel Malik et al., 2003 [123]	Retrospective case-control study, $n = 67$ people with schizophrenia or schizo-affective disorder matched with 102 unaffected siblings in families with multiple occurrences of schizophrenia	3	Childhood head injuries occurred in 24% of schizophrenia patients versus 12% of unaffected siblings Schizophrenia cases had an excess of childhood head injuries, with an OR of 2.34 for head injuries before the age of 10, and an OR of 1.90 for head injuries occurring up to the age of 17 years
Thomsen et al., 1984 [124]	$n = 40$ severe TBI, 10–15 years follow-up	4	Posttraumatic psychoses: 20%

TBI: traumatic brain injury.

Table 7
Main studies on prevalence rates of suicides and suicide attempts after traumatic brain injury.

References	Design	Level	Conclusion
Silver et al., 2001 [111]	Cohort study: Prevalence rates of neuropsychiatric disorder and suicide in individuals with and without a history of traumatic brain injury (New Haven NIMH Epidemiological Catchment Area Study), $n = 5037$	2	Tentative attempts with TBI: RR = 4
Tsaousides et al., 2011 [131]	Cohort study: prevalence rates of suicidal ideation in a sample of community-dwelling adults with mild to severe traumatic brain injury, $n = 356$ TBI	2	The rate of SI was 28% Premorbid substance abuse, psychiatric diagnosis of depression, anxiety, or posttraumatic stress disorder are risk factors. SI are significantly correlated with lower psychosocial functioning
Tate, 1997 [132]	Retrospective study: premorbid psychosocial variables, injury details, and posttraumatic sequelae of eight completed suicides after TBI. $n = 896$ severe TBI admitted into a regional brain injury rehabilitation service over a period of 18 years	4	8 suicides among 50 deaths 7/8 with suicide ideations before suicide Suicide attempts: 60% before suicide
Teasdale and Endberg, 2001 [41]	Archival study: prevalence of suicide among patients with TBI in Denmark, $n = 145{,}000$	4	Suicide: 0.6% for patients with cranial fracture ($n = 46/7560$), 0.8% for patient with a cerebral contusion or traumatic intracranial haemorrhage ($n = 99/11{,}766$) The incidence of suicide was increased relative to the general population (respectively 2.7 after cranial fracture, and 4.1 after cerebral contusion) The ratios were higher for females than for males, with substance misuse and lower for patients injured before the age of 21 or after the age of 60
Simpson et al., 2002 [128]	Prevalence of hopelessness, suicidal ideation and suicide attempts after TBI, $n = 172$ TBI	4	Levels of hopelessness (35%) Suicide ideation (23%) Suicide attempt post-injury (18%)
Simpson et al., 2005 [129]	Retrospective study, $n = 172$ TBI	4	45 clients (26.2%) had collectively made a lifetime total of 80 suicide attempts. The sample had a lifetime prevalence of 1.86 attempts, with 19 clients (44.2%) making repeat attempts. Attempts post-TBI with a comorbid post-injury history of psychiatric/emotional disturbance and substance abuse. OR = 21
Harrison-Felix et al., 2009 [133]	Retrospective cohort study, $n = 1678$ TBI admitted to the rehabilitative Craig Hospital	4	18% expired due to external causes of injury, half from suicide

TBI: traumatic brain injury; SI: suicide ideations.

disruptive primary behaviors by default, affective disorders – anxiety – psychosis and suicide attempts and suicide. Furthermore, research studies did not focus on theory of mind or social cognition impairments, even if recent works have developed these research pathways. Psychological disorders affecting the subjective self-identity, perfectly explained by Oppenheim-Gluckman [134] or personality were not treated either, even if all these cognitive, psychological or somatic facets are intertwined and it is necessary to take them into account.

The issue of "behavioral disorder" is complex, it is located at the boundaries of neurology, neuropsychology, physical medicine and rehabilitation (PM&R) and psychiatry. Older and more recent psychiatric classifications (CIM 10, DSM-IV and now DSM 5) report limits in the description and classification of TBI-related disorders, the use of specific scales tailored to patients with TBI seems more adapted [135]. The notion of behavior by excess and behavior by default meets the description of Levin et al. in 1991 [136] and Luria in 1966 [137] who identified an impairment mode in case of affection of the frontal convexity (initiative, planning deficits, judgment disorders, egocentricity) and in case of orbitofrontal damage emotional control deficits with disinhibition, irritability, agitation, and loss of social norms. One can note that the Behavioral Dysexecutive Syndrome Inventory scale (Inventaire du Syndrome Dysexécutif Comportemental, ISDC) developed by the GREFEX takes back the classification of global under- and over-activity for self-initiated and hetero-initiated activities, which is very similar to the nomenclature we are proposing [138].

The objective of this work was to refine the prevalence of a symptom, but obviously several symptoms can be expressed at the same time or successively. Thus, major depression is frequently associated with posttraumatic aggression [19,25,34]. In this work, all symptoms presented, except for substance abuse, showed higher prevalence rates than in the general population. Major personality changes, loss of tolerance to frustration, sustainable anxiety, and anger or aggression manifestations were observed 2 to 10 years posttrauma [139]. In 1995, Ponsford et al. [139] evaluated, via a behavioral questionnaire, 175 patients 2 years after traumatic brain injury. Irritability/aggression was the most common symptom (67%) before loss of initiative (44%), impulsiveness (43%), self-centred behavior (28%) or inappropriate social behavior (26%). Mood disorders (anxiety and depression) were present in more than half the cases. The aggravation over time of these manifestations, negatively impacts family distress and adds to the patient's stress [3]. Aggressive behaviors can be at the forefront [15,140], sometimes aggravated by mood swings [25] but on the longer term, disruptive primary behaviors by excess (aggression, disinhibition, agitation, mental excitability) seem less frequent than behavioral changes by default (decreased initiative, isolation, withdrawal) [124]. Depressive symptoms and anxious disorders remain present a long time after the trauma, up to 52% and 35% of cases respectively at 5 years in the epidemiology study conducted in the French region of Aquitaine (Evaluation with the NRS-R scale). They are considered the most debilitating sequelae at a distance from the trauma for families and negatively impacting the patient's reintegration into social life [141].

The work here focuses on moderate to severe TBI. Studies found often combined mild, moderate and severe TBI. It was sometimes difficult to analyze the results of these studies that did not differentiate the initial severity of the trauma or that did not give a precise enough definition of the trauma. It appears that the prevalence of disorders is not the same depending on the initial severity of the trauma [1]. For example, patients with severe TBI seem to present more irritability than patients with mild TBI but not depression. Similarly, studies on war veterans, which might not be applicable to civilians with TBI, were not kept for this work. Finally, most studies did not indicate the status of the patient and personality pre-TBI, limiting the accountability of TBI in the onset of symptoms. Nevertheless, the individual and social impact of these behavioral disorders, like their magnitude, participates to the "severity" of the traumatic brain injury. In this heterogeneous population, one of the objectives to be reached is to develop long-term cohort studies. Several studies are in fact already underway (ESPARR study [142] and PariS-TBI Study [143]).

5. Conclusion

The development of good practices recommendation regarding the care management of behavioral disorders after traumatic brain injury is based first on clarifying the nomenclature and highlighting the rather high prevalence of symptoms, and their sustainability. Traumatic brain injury must be studied in its somatic, neuropsychological, psychological, psychiatric dimensions while assessing the psycho-dynamics adjustments of an individual with a basic personality, in a disrupted environmental context.

The French Society of Physical and Rehabilitation Medicine (SOFMER) Group

Mission French High Authority for Health (HAS) Officers

Dr. Muriel Dhenain, HAS, project manager; Dr. Philippe Blanchard, HAS, project manager; Mrs. Emmanuelle Blondet, HAS, librarian.

Steering group

Pr. Jean-François Mathé, Physical Medicine and Rehabilitation physician (PM&R) – Chairman of the steering group; Dr. Jean-Jacques Dumond, psychiatrist; Mr. Emeric Guillermou, Lawyer, representing the families, National Union of Traumatic Brain Injury Families (UNAFTC) President; Pr. Jean-Michel Mazaux, PM&R physician; Mr. Michel Onillon, Branch Manager; Pr. Pradat-Diehl Pascale, PM&R physician.

Working group French Society of Physical Medicine and Rehabilitation (SOFMER)

Pr. Jacques Luauté, PM&R physician – Chairman of the working group; Dr. Julia Hamonet, PM&R physician, project manager; Dr. David Plantier, PM&R physician, project manager; Dr. Stefan Angelique PM&R physician, project manager; Dr. Laurent Wiart, PM&R physician, project manager; Mrs. Annabelle Arnould, psychologist; Mrs. Suzanne Aubert, representing the families UNAFTC; Dr. Jean-Marie Beis, PM&R physician; Mr. Laurent Blais, Director, nursing home; Mrs. Marie-Christine Cazals, representing the families UNAFTC; Dr. Jean-Marc Destaillats, psychiatrist; Dr. Eric Durand, PM&R physician; Dr. Patrick Fayol, psychiatrist; Dr. Christine Fieyre, general practitioner (Departmental Home for Disabled Persons); Mr. Luc Jagot, psychologist; Dr. Christophe Lermuzeaux, psychiatrist; Mr. Jean-Michel Lucas, professor of physical education and sports; Dr. Dominique Malauzat, psychiatrist, pharmacologist; Mrs. Nelly Montrobert, social worker; Mr. Jacques Antoine Preziosi, Lawyer; Mrs. Antoinette Prouteau, psychologist; Pr. Isabelle Richard, PM&R physician; Dr. Tell Laurence, PM&R physician.

Reading group

Professor Philippe Allain, psychologist; Dr. Laurent Atlani, PM&R physician; Pr. Philippe Azouvi, PM&R physician; Dr. Eleonore Bayen, PM&R physician; Mr. Christian Belio, occupational therapist; Master Richard Bometon, Magistrate; Mrs. Céline Bonnyaud, physiotherapist; Mr. Marc Ceccaldi, Lawyer; Mrs. Renée Chaignon, social worker; Dr. Emmanuel Chevrillon, PM&R physician; Mrs. Dominique Chopinaud, care settings; Mrs. Christine Croisiaux, psychologist, European Brain Injury Society (EBIS) President; Dr. Xavier Debelleix, PM&R physician; Mrs. Brigitte Dherbey, representing the families, UNAFTC; Mr. Philippe Hingray Insurance MAAF; Dr. Corinne Jockic, PM&R physician; Mrs. Françoise Joyeux, Psychologist; Dr. Françoise Laloua, PM&R physician; Pr. Didier Le Gall, psychologist; Mrs. Jacqueline Madinier, representing the families, UNAFTC; Mrs. Anne-Cécile Marquet, nurse; Pr. Michèle Montreuil, psychologist; Dr. Hélène Oppenheim-Gluckman, psychiatrist and psychoanalyst; Mrs. Annie Perussel, Director of an establishment; Dr Bruno Pollez, PM&R physician; Mr. Stéphane Raffard, psychologist; Dr. André-Jean Remy, doctor hepatologist – prison; Dr. Marc Rousseau, PM&R physician; Mrs. Véronique Roussenac, Psychologist; Dr. Virginie Saout, PM&R physician; Pr Jean-Luc Truelle, neurologist; Pr Yves Zerbib, general practitioner.

Disclosure of interest

The authors declare that they have no competing interest.

Acknowledgements

The authors would like to thank the SOFMER at the origin of this work and the French Traumatic Brain Injury (France Traumatisme Crânien) association for their financial support (reimbursement of travel fees) and the French High Authority for Health (HAS) for lending them space and for their help with the literature research. Experts did not receive financial retribution for their work.

References

[1] Deb S, Lyons I, Koutzoukis C. Neurobehavioural symptoms one year after a head injury. Br J Psychiatry 1999;174:360–5.
[2] Masson F, Maurette P, Salmi LR, Dartigues JF, Vecsey J, Destaillats JM, et al. Prevalence of impairments 5 years after a head injury, and their relationship with disabilities and outcome. Brain Inj 1996;10:487–97.
[3] Brooks DN, Campsie L, Symington C. The five-year outcome of severe blunt head injury: a relative's view. J Neurol Neurosurg Psychiatry 1986;49:764–70.
[4] Ponsford JL, Olver JH, Curran C, et al. Prediction of employment status 2 years after traumatic brain injury. Brain Inj 1995;9:11–20.
[5] Tassé MJ, Sabourin G, Garcin N, Lecavalier L. Définition d'un trouble grave du comportement chez les personnes ayant une déficience intellectuelle. Can J Behav Sci 2010;42:62–9.
[6] Hutton B, Salanti G, Caldwell DM, Chaimani A, Schmid CH, Cameron C, et al. The PRISMA extension statement for reporting of systematic reviews incorporating network meta-analyses of health care interventions: checklist and explanations. Ann Intern Med 2015;162:777–84.
[7] Hagen C, Malkmus D, Durham P. Levels of cognitive functioning. In: Presented at 3rd Annual Post-Graduate Course on rehabilitation of traumatic brain-injured Adult; 1979.p. 14–6.
[8] Bogner J, Corrigan JD. Epidemiology of agitation following brain injury. Neurorehabilitation 1995;5:293–7.
[9] Sandel ME, Mysiw WJ. The agitated brain-injured patient. Part 1: definitions, differential diagnosis, and assessment. Arch Phys Med Rehabil 1996;77:617–23.

[10] Levin HS, Grossman RG. Behavioral sequelae of closed head injury: a quantitative study. Arch Neurol 1978;35:720–7.
[11] Reyes RL, Bhattacharyya AK, Heller D. Traumatic head injury: restlessness and agitation as prognosticators of physical and psychologic improvement in patients. Arch Phys Med Rehabil 1981;62:20–3.
[12] Fugate LP, Spacek LA, Kresty LA, Levy CE, Johnson JC, Mysiw WJ. Measurement and treatment of agitation following traumatic brain injury: II. A survey of the Brain Injury Special Interest Group of the American Academy of Physical Medicine and Rehabilitation. Arch Phys Med Rehabil 1997;78:924–8.
[13] Lombard LA, Zafonte RD. Agitation after traumatic brain injury: considerations and treatment options. Am J Phys Med Rehabil 2005;84:797–812.
[14] Singh R, Venkateshwara G, Nair KP, Khan M, Saad R. Agitation after traumatic brain injury and predictors of outcome. Brain Inj 2014;28:336–40.
[15] Brooke MM, Questad KA, Patterson DR, Bashak KJ. Agitation and restlessness after closed head injury: a prospective study of 100 consecutive admissions. Arch Phys Med Rehabil 1992;73:320–3.
[16] Wolf AP, Gleckman AD, Cifu DX, Ginsburg PC. The prevalence of agitation and brain injury in skilled nursing facilities: a survey. Brain Inj 1996;10:241–5.
[17] Bogner JA, Corrigan JD, Fugate L, Mysiw WJ, Clinchot D. Role of agitation in prediction of outcomes after traumatic brain injury. Am J Phys Med Rehabil 2001;80:636–44.
[18] Nott MT, Chapparo C, Baguley IJ. Agitation following traumatic brain injury: an Australian sample. Brain Inj 2006;20:1175–82.
[19] Yudofsky SC, Silver JM, JacksonW, Endicott J, Williams D. The Overt Aggression Scale for the objective rating of verbal and physical aggression. Am J Psychiatry 1986;143:35–9.
[20] Fleminger S, Greenwood RRJ, Oliver DL. Pharmacological management for agitation and aggression in people with acquired brain injury. Cochrane Database Syst Rev 2008;18. CD003299.
[21] Silver JM, Yudofsky SC. Aggressive disorders. In: Silver JM, Yudofsky SC, Hales RE, editors. Neuropsychiatry of traumatic brain injury. Washington DC: American Psychiatric Press; 1994. p. 313–53.
[22] Rapoport M, McCauley S, Levin H, Song J, Feinstein A. The role of injury severity in neurobehavioral outcome 3 months after traumatic brain injury. Neuropsychiatry Neuropsychol Behav Neurol 2002;15:123–32.
[23] Tateno A, Jorge RE, Robinson RG. Clinical correlates of aggressive behavior after traumatic brain injury. J Neuropsychiatry Clin Neurosci 2003;15:155–60.
[24] Alderman N. Prevalence, characteristics and causes of aggressive behaviour observed within a neurobehavioural rehabilitation service: predictors and implications for management. Brain Inj 2007;21:891–911.
[25] Baguley IJ, Cooper J, Felmingham K. Aggressive behavior following traumatic brain injury: how common is common? J Head Trauma Rehabil 2006;21:45–56.
[26] McDonald S, Hunt C, Henry JD, Dimoska A, Bornhofen C. Angry responses to emotional events: the role of impaired control and drive in people with severe traumatic brain injury. J Clin Exp Neuropsychol 2010;32:855–64.
[27] James AL, Young AW. Clinical correlates of verbal aggression, physical aggression and inappropriate sexual behaviour after brain injury. Brain Inj 2013;27:1162–72.
[28] Arciniegas DB, Wortzel HS. Emotional and behavioral dyscontrol after traumatic brain injury. Psychiatr Clin North Am 2014;37:31–53.
[29] Galski T, Palasz J, Bruno RL, Walker JE. Predicting physical and verbal aggression on a brain trauma unit. Arch Phys Med Rehabil 1994;75:380–3.
[30] Johnson R, Balleny H. Behaviour problems after brain injury: incidence and need for treatment. Clin Rehabil 1996;10:173–80.
[31] Alderman N, Knight C, Morgan C. Use of a modified version of the Overt Aggression Scale in the measurement and assessment of aggressive behaviours following brain injury. Brain Inj 1997;11:503–23.
[32] Kant R, Smith-Seemiller L, Zeiler D. Treatment of aggression and irritability after head injury. Brain Inj 1998;12:661–6.
[33] Giles GM, Mohr JD. Overview and inter-rater reliability of an incident-based rating scale for aggressive behaviour following traumatic brain injury: the Overt Aggression Scale-Modified for Neurorehabilitation-Extended (OAS-MNR-E). Brain Inj 2007;21:505–11.
[34] Rao V, Rosenberg P, Bertrand M, Salehinia S, Spiro J, Vaishnavi S, et al. Aggression after traumatic brain injury: prevalence and correlates. J Neuropsychiatry Clin Neurosci 2009;21:420–9.
[35] Dickens G, Alderman N, Bowers L. Potential severity of aggressive behaviour after acquired brain injury: implications for recording. J Psychiatr Ment Health Nurs 2011;18:586–94.
[36] Safer J. Irritable mood and the diagnosis and Statistical Manual of Mental Disorders. Child Adolesc Psychiatry Ment Health 2009;3:35.
[37] Yang CC, Hua MS, Lin WC, Tsai YH, Huang SJ. Irritability following traumatic brain injury: divergent manifestations of annoyance and verbal aggression. Brain Inj 2012;26:1185–91.
[38] Yang CC, Huang SJ, Lin WC, Tsai YH, Hua MS. Divergent manifestations of irritability in patients with mild and moderate to severe traumatic brain injury: perspectives of awareness and neurocognitive correlates. Brain Inj 2013;27:1008–15.
[39] Ponsford J, Whelan-Goodinson R, Bahar-Fuchs A. Alcohol and drug use following traumatic brain injury: a prospective study. Brain Inj 2007;21:1385–92.
[40] Reslan S, Hanks RA. Factors associated with alcohol-related problems following moderate to severe traumatic brain injury. Rehabil Psychol 2014;59:453–8.
[41] Graham DP, Cardon AL. An update on substance use and treatment following traumatic brain injury. Ann N Y Acad Sci 2008;1141:148–62.
[42] Teasdale TW, Engberg A. Suicide after traumatic brain injury: a population study. J Neurol Neurosurg Psychiatry 2001;71:436–40.
[43] Castaño B, Capdevila E. Eating disorders in patients with traumatic brain injury: a report of four cases. NeuroRehabilitation 2010;27:113–6.
[44] Saoût V, Gambart G, Leguay D, Ferrapie AL, Launay C, Richard I. Aggressive behavior after traumatic brain injury. Ann Phys Rehabil Med 2011;54:259–69.
[45] Richard I, Rome J, Lemene B, Louis F, Perrouin-Verbe B, Mathe JF. Déficits endocriniens post-traumatiques : analyse d'une série de 93 traumatismes crâniens graves. Ann Phys Rehabil Med 2001;4:19–25.
[46] Farrer TJ, Hedges DW. Prevalence of traumatic brain injury in incarcerated groups compared to the general population: a meta-analysis. Prog Neuropsychopharmacol Biol Psychiatry 2011;35:390–4.
[47] Shiroma EJ, Ferguson PL, Pickelsimer EE. Prevalence of traumatic brain injury in an offender population: a meta-analysis. J Correct Health Care 2010;16:147–59.
[48] Starkstein SE, Pahissa J. Apathy following traumatic brain injury. Psychiatr Clin North Am 2014;37:103–12.
[49] Marin RS, Biedrzycki RC, Firinciogullari S. Reliability and validity of the Apathy Evaluation Scale. Psychiatry Res 1991;38:143–62.
[50] Levy R, Dubois B. Apathy and the functional anatomy of the prefrontal cortex-basal ganglia circuits. Cerebral Cortex 2006;16:916–28.
[51] Robert PH, Onyike CU, Leentjens AF, Dujardin K, Aalten P, Starkstein S, et al. Proposed diagnostic criteria for apathy in Alzheimer's disease and other neuropsychiatric disorders. Eur Psychiatry 2009;24:98–104.
[52] Mulin E, Leone E, Dujardin K, Delliaux M, Leentjens A, Nobili F, et al. Diagnostic criteria for apathy in clinical practice. Int J Geriatr Psychiatry 2011;26:158–65.
[53] Kant R, Duffy JD, Pivovarnik A. Prevalence of apathy following head injury. Brain Inj 1998;12:87–92.
[54] Rao V, Spiro JR, Schretlen DJ, Cascella NG. Apathy syndrome after traumatic brain injury compared with deficits in schizophrenia. Psychosomatics 2007;48:217–22.
[55] Andersson S, Bergedalen AM. Cognitive correlates of apathy in traumatic brain injury. Neuropsychiatry Neuropsychol Behav Neurol 2002;15:184–91.
[56] Muller U, Czymmek J, Thone-Otto A, Von Cramon DY. Reduced daytime activity in patients with acquired brain damage and apathy: a study with ambulatory actigraphy. Brain Inj 2006;20:157–60.
[57] Andersson S, Gundersen S, Finset A. Emotional activation during therapeutic interaction in traumatic brain injury: effect of apathy, self-awareness and implications for rehabilitation. Brain Inj 1999;13:393–404.
[58] Al-Adawi S, Dorvlo AS, Burke DT, Huynh CC, Jacob L, Knight R, et al. Apathy and depression in cross-cultural survivors of traumatic brain injury. J Neuropsychiatry Clin Neurosci 2004;16:435–42.
[59] Lane-Brown AT, Tate RL. Measuring apathy after traumatic brain injury: Psychometric properties of the Apathy Evaluation Scale and the Frontal Systems Behavior Scale. Brain Inj 2009;23:999–1007.
[60] Ciurli P, Formisano R, Bivona U, Cantagallo A, Angelelli P. Neuropsychiatric disorders in persons with severe traumatic brain injury: prevalence, phenomenology, and relationship with demographic, clinical, and functional features. J Head Trauma Rehabil 2011;26:116–26.
[61] Jorge RE, Arciniegas DB. Mood disorders after TBI. Psychiatr Clin North Am 2014;37:13–29.
[62] Kim E, Lauterbach EC, Reeve A, Arciniegas DB, Coburn KL, Mendez MF, et al. Neuropsychiatric complications of traumatic brain injury: a critical review of the literature (a report by the ANPA Committee on Research). J Neuropsychiatry Clin Neurosci 2007;19:106–27.
[63] Van Reekum R, Cohen T, Wong J. Can traumatic brain injury cause psychiatric disorders? J Neuropsychiatry Clin Neurosci 2000;12:316–27.
[64] Lermuzeaux C. Les troubles psychiatriques post-traumatiques chez le traumatise crânien. Info Psychiatr 2012;88:345–52.
[65] Jorge RE, Robinson RG, Arndt SV, et al. Depression following traumatic brain injury: a 1-year longitudinal study. J Affect Disord 1993;27:233–43.
[66] Valk-Kleibeuker L, Heijenbrok-Kal MH, Ribbers GM. Mood after moderate and severe traumatic brain injury: a prospective cohort study. PLoS One 2014;4:e87414.
[67] Bombardier CH, Fann JR, Temkin NR, et al. Rates of major depressive disorder and clinical outcomes following traumatic brain injury. JAMA 2010;303:1938–45.
[68] Hart T, Brenner L, Clark AN, Bogner JA, Novack TA, Chervoneva I, et al. Major and minor depression after traumatic brain injury. Arch Phys Med Rehabil 2011;92:1211–9.
[69] Dunlop TW, Udvarhelyi GB, Stedem AF, et al. Comparison of patients with and without emotional/behavioral deterioration during the first year after traumatic brain injury. J Neuropsychiatry Clin Neurosci 1991;3:150–6.
[70] McCleary C, Satz P, Forney D, et al. Depression after traumatic brain injury as a function of Glasgow Outcome Score. J Clin Exp Neuropsychol 1998;20:270–9.
[71] Curran CA, Ponsford JL, Crowe S. Coping strategies and emotional outcome following traumatic brain injury: a comparison with orthopedic patients. J Head Trauma Rehabil 2000;15:1256–74.
[72] Fann JR, Burington B, Leonetti A, Jaffe K, Katon W, Thompson R. Psychiatric illness following traumatic brain injury in an adult health maintenance organization population. Arch Gen Psychiatry 2004;61:53–61.

[73] Jorge RE, Robinson RG, Moser D, et al. Major depression following traumatic brain injury. Arch Gen Psychiatry 2004;61:42–50.
[74] Popovic V, Pekic S, Pavlovic D, et al. Hypopituitarism as a consequence of traumatic brain injury (TBI) and its possible relation with cognitive disabilities and mental distress. J Endocrinol Invest 2004;27:1048–54.
[75] Frenisy MC, Benony H, Chahraoui K, et al. Brain-injured patients versus multiple trauma patients: some neurobehavioral and psychopathological aspects. J Trauma 2006;60:1018–26.
[76] Gagnon J, Bouchard MA, Rainville C, et al. Inhibition and object relations in borderline personality traits after traumatic brain injury. Brain Inj 2006;20: 67–81.
[77] Ziino C, Ponsford J. Selective attention deficits and subjective fatigue following traumatic brain injury. Neuropsychology 2006;20:383–90.
[78] Hawthorne G, Gruen RL, Kaye AH. Traumatic brain injury and long-term quality of life: findings from an Australian study. J Neurotrauma 2009;26: 1623–33.
[79] Varney NR, Martzke JS, Roberts RJ. Major depression in patients with closed head injury. Neuropsychology 1987;1:7–9.
[80] Fann JR, Katon WJ, Uomoto JM, et al. Psychiatric disorders and functional disability in outpatients with traumatic brain injuries. Am J Psychiatry 1995;152:1493–9.
[81] Gomez-Hernandez R, Max JE, Kosier T, et al. Social impairment and depression after traumatic brain injury. Arch Phys Med Rehabil 1997;78: 1321–6.
[82] Deb S, Lyons I, Koutzoukis C, Ali I, McCarthy G. Rate of psychiatric illness 1 year after traumatic brain injury. Am J Psychiatry 1999;156:374–8.
[83] Sherman EM, Strauss E, Slick DJ, et al. Effect of depression on neuropsychological functioning in head injury: measurable but minimal. Brain Inj 2000; 14:621–32.
[84] Bryant RA, Marosszeky JE, Crooks J, et al. Posttraumatic stress disorder and psychosocial functioning after severe traumatic brain injury. J Nerv Ment Dis 2001;189:109–13.
[85] Kersel DA, Marsh NV, Havill JH, et al. Psychosocial functioning during the year following severe traumatic brain injury. Brain Inj 2001;15:683–96.
[86] Seel RT, Kreutzer JS. Depression assessment after traumatic brain injury: an empirically based classification method. Arch Phys Med Rehabil 2003;84: 1621–8.
[87] Ashman TA, Spielman LA, Hibbard MR, et al. Psychiatric challenges in the first 6 years after traumatic brain injury: cross-sequential analyses of Axis I disorders. Arch Phys Med Rehabil 2004;85:S36–42.
[88] Franulic A, Carbonell CG, Pinto P, et al. Psychosocial adjustment and employment outcome 2, 5 and 10 years after TBI. Brain Inj 2004;18:119–29.
[89] Evans CC, Sherer M, Nick TG, et al. Early impaired self-awareness, depression, and subjective well-being following traumatic brain injury. J Head Trauma Rehabil 2005;20:488–500.
[90] Huang DB, Spiga R, Koo H. Use of the Zung depression scale in patients with traumatic brain injury: 1 year post-injury. Brain Inj 2005;19:903–8.
[91] Kennedy RE, Livingston L, Riddick A, et al. Evaluation of the Neurobehavioral Functioning Inventory as a depression screening tool after traumatic brain injury. J Head Trauma Rehabil 2005;20:512–26.
[92] Chiu WT, Huang SJ, Hwang HF, et al. Use of the WHOQOL-BREF for evaluating persons with traumatic brain injury. J Neurotrauma 2006;23:1609–20.
[93] Rapoport MJ, Herrmann N, Shammi P, et al. Outcome after traumatic brain injury sustained in older adulthood: a one-year longitudinal study. Am J Geriatr Psychiatry 2006;14:456–65.
[94] Sherer M, Evans CC, Leverenz J, et al. Therapeutic alliance in post-acute brain injury rehabilitation: predictors of strength of alliance and impact of alliance on outcome. Brain Inj 2007;21:663–72 [52].
[95] Al-Adawi S, Dorvlo AS, Al-Naamani A, et al. The ineffectiveness of the Hospital Anxiety and Depression Scale for diagnosis in an Omani traumatic brain injured population. Brain Inj 2007;21:385–93.
[96] Hawley CA, Joseph S. Predictors of positive growth after traumatic brain injury: a longitudinal study. Brain Inj 2008;22:427–35.
[97] Whelan-Goodinson R, Ponsford J, Schonberger M. Association between psychiatric state and outcome following traumatic brain injury. J Rehabil Med 2008;40:850–7.
[98] Fann JR, Jones AL, Dikmen SS, et al. Depression treatment preferences after traumatic brain injury. J Head Trauma Rehabil 2009;24:272–8.
[99] Peleg G, Barak O, Harel Y, et al. Hope, dispositional optimism and severity of depression following traumatic brain injury. Brain Injury 2009;23: 800–8.
[100] Ponsford J, Schönberger M. Family functioning and emotional state two and five years after traumatic brain injury. J Int Neuropsychol Soc 2010; 16:306–17.
[101] Seel RT, Macciocchi S, Kreutzer JS. Clinical considerations for the diagnosis of major depression after moderate to severe TBI. J Head Trauma Rehabil 2010;25:99–112.
[102] Van Reekum R, Bolago I, Finlayson MA, et al. Psychiatric disorders after traumatic brain injury. Brain Inj 1996;10:319–27.
[103] Rueckriegel SM, Baron M, Domschke K, Neuderth S, Kunze E, Kessler AF, et al. Trauma- and distress-associated mental illness symptoms in close relatives of patients with severe traumatic brain injury and high-grade subarachnoid hemorrhage. Acta Neurochir 2015;157:1329–36.
[104] Alway Y, McKay A, Gould KR, Johnston L, Ponsford J. Factors associated with posttraumaic stress disorder following moderate to severe traumatic brain injury: a prospective study. Depress Anxiety 2015;28.
[105] Sigurdardottir S, Andelic N, Roe C, Schanke AK. Identifying longitudinal trajectories of emotional distress symptoms 5 years after traumatic brain injury. Brain Inj 2014;28:1542–50.
[106] Bahraini NH, Breshears RE, Hernández TD, Schneider AL, Forster JE, Brenner LA. Traumatic brain injury and posttraumatic stress disorder. Psychiatr Clin North Am 2014;37:55–75.
[107] Kant R, Smith-Seemiller L, Duffy JD. Obsessive-compulsive disorder after closed head injury: review of literature and report of four cases. Brain Inj 1996;10:55–63.
[108] Berthier ML, Kulisevsky JJ, Gironell A, Lopez OL. Obsessive-compulsive disorder and traumatic brain injury: behavioral, cognitive, and neuroimaging findings. Neuropsychiatry Neuropsychol Behav Neurol 2001;14:23–31.
[109] Hibbard MR, Uysal S, Kepler K, Bogdany J, Silver J. 'Axis I psychopathology in individuals with traumatic brain injury'. J Head Trauma Rehabil 1998; 13:24–39.
[110] Hoofien D, Gilboa A, Vakil E, Donovick PJ. Traumatic brain injury (TBI) 10–20 years later: a comprehensive outcome study of psychiatric symptomatology, cognitive habilities and psychosocial functioning. Brain Inj 2001;15: 935–45.
[111] Silver JM, Kramer R, Greenwald S, Weissman M. 'The association between head injuries and psychiatric disorders: findings from the New Haven NIMH Epidemiologic Catchment Area Study'. Brain Inj 2001;15:935–45.
[112] Koponen S, Taiminen T, Portin R, Himanen L, Isoniemi H, Heinonen H, et al. Axis I and II psychiatric disorders after traumatic brain injury: a 30-year follow-up study. Am J Psychiatry 2002;159:1315–21.
[113] Hesdorffer DC, Rauch SL, Tamminga CA. Long-term psychiatric outcomes following traumatic brain injury: a review of the literature. J Head Trauma Rehabil 2009;24:452–9.
[114] Molloy C, Conroy RM, Cotter DR, Cannon M. Is traumatic brain injury a risk factor for schizophrenia?. A meta-analysis of case-controlled population-based studies. Schizophr Bull 2011;37:1104–10.
[115] David A, Prince M. Psychosis following head injury: a critical review. J Neurol Neurosurg Psychiatry 2005;76:153–60.
[116] Hang RH, Xu YJ, Zhu XY. Correlative factors for organic psychotic symptoms in patients following traumatic brain injury. Fa Yi Xue Za Zhi 2014;30:36–40.
[117] Kim E. Does TBI predispose individuals to develop schizophrenia? Curr Opin Psychiatry 2008;21:286–9.
[118] Malaspina D, Goetz RR, Friedman JH, et al. Traumatic brain injury and schizophrenia in members of schizophrenia and bipolar disorder pedigrees. Am J Psychiatry 2001;158:440–6.
[119] Timonen M, Miettunen J, Hakko H, et al. The association of preceding traumatic brain injury with mental disorders, alcoholism and criminality: the Northern Finland 1966 Birth Cohort Study. Psychiatry Res 2002;113: 217–26.
[120] Harrison G, Whitley E, Rasmussen F, Lewis G, Dalman C, Gunnell D. Risk of schizophrenia and other non-affective psychosis among individuals exposed to head injury: case-control study. Schizophr Res 2006;88:119–26.
[121] Sachdev P, Smith JS, Cathcart S. Schizophrenia-like psychosis following traumatic brain injury: a chart-based descriptive and case-control study. Psychol Med 2001;31:231–9.
[122] Nielsen AS, Mortensen PB, O'Callaghan E, Mors O, Ewald H. Is head injury a risk factor for schizophrenia? Schizophr Res 2002;55:93–8.
[123] Abdel Malik P, Husted J, Chow EW, Bassett AS. Childhood head injury and expression of schizophrenia in multiply affected families. Arch Gen Psychiatry 2003;60:231–6.
[124] Thomsen IV. Late outcome of very severe blunt head trauma: a 10–15 years second follow-up. J Neurol Neurosurg Psychiatry 1984;47:260–8.
[125] Simpson G, Tate R. Suicidality in people surviving a traumatic brain injury: prevalence, risk factors and implications for clinical management. Brain Inj 2007;21:1335–51.
[126] Fujii DE, Ahmed I. Psychotic disorder caused by traumatic brain injury. Psychiatr Clin North Am 2014;37:113–24.
[127] Fazel S, Wolf A, Pillas D, Lichtenstein P, Långström N. Suicide, fatal injuries, and other causes of premature mortality in patients with traumatic brain injury: a 41-year Swedish population study. JAMA Psychiatry 2014;71: 326–33.
[128] Simpson GK, Tate RL. Suicidality after traumatic brain injury: demographics, injury and clinical correlates. Psychol Med 2002;32:687–97.
[129] Simpson G, Tate R. Clinical features of suicide attempts after traumatic brain injury. J Nerv Mental Dis 2005;193:680–5.
[130] Mackelprang JL, Bombardier CH, Fann JR, Temkin NR, Barber JK, Dikmen SS. Rates and predictors of suicidal ideation during the first year after traumatic brain injury. Am J Public Health 2014;104:e100–7.
[131] Tsaousides T, Cantor JB, Gordon WA. Suicidal ideation following traumatic brain injury: prevalence rates and correlates in adults living in the community. J Head Trauma Rehabil 2011;26:265–75.
[132] Tate RL, Simpson GK, Flanagan S, Coffey M. Completed suicide after traumatic brain injury. J Head Trauma Rehabil 1997;12:16–28.
[133] Harrison-Felix CL, Whiteneck GG, Jha A, et al. Mortality over four decades after traumatic brain injury rehabilitation: a retrospective cohort study. Arch Phys Med Rehabil 2009;90:1506–13.
[134] Oppenheim-Gluckman H. La pensée naufragée. Clinique psychopathologique des patients cérébrolésés. Édition Economica Anthropos; 2006.
[135] Fayol P, Dumond JJ. Les limites de la nosographie psychiatrique dans les troubles psychiques des traumatisés crâniens sévères. Ann Readapt Med Phys 1997;40:543–6.

[136] Levin, Heiseberg HM, Benton AL, editors. Frontal lobe function and dysfunction. New York: Oxford University Press; 1991 [427 p.].
[137] Luria AR. Higher cortical functions in man. New York, NY: Basic Books; 1966.
[138] Godefroy O, Azouvi P, Robert P, Roussel M, LeGall D, Meulemans T, et al. Dysexecutive syndrome: diagnostic criteria and validation study. Ann Neurol 2010;68:855–64.
[139] Ponsford JL, Olver JH, Curran C. A profile of outcome: 2 years after traumatic brain injury. Brain Inj 1995;9:1–10.
[140] DeGuise E, LeBlanc J, Feyz M, Meyer K, Duplantie J, Thomas H, et al. Long-term outcome after severe traumatic brain injury: the McGill interdisciplinary prospective study. J Head Trauma Rehabil 2008;23:294–303.
[141] Alaoui P, Mazaux JM, Masson F, Vecsey J, Destaillats JM, Maurette P, et al. Devenir neuropsychologique à long terme des traumatisés crâniens. Évaluation à cinq ans des troubles neuropsychologiques et comportementaux par l'Échelle Neurocomportementale Révisée (à propos de 79 cas). Ann Readapt Med Phys 1998;41:171–81.
[142] Nash S, Luauté J, Bar JY, Sancho PO, Hours M, Chossegros L, et al. Cognitive and behavioural posttraumatic impairments: what is the specificity of a brain injury? A study within the ESPARR cohort. Ann Phys Rehabil Med 2014;57:600–17.
[143] Jourdan C, Bosserelle V, Azerad S, Ghout I, Bayen E, Aegerter P, et al. Predictive factors for 1-year outcome of a cohort of patients with severe traumatic brain injury (TBI): results from the PariS-TBI study. Brain Inj 2013;27:1000–7.